imparted by this one line entry makes the Examiner's comments invalid. The rejections are transversed and Claims 3, 4, 8, 23 and 24 are retained.

In consideration of the Point 2, we suggest a new Main Claim that would replace these five claims (see Claim 25 in Current Claims List). The new claim addresses the conflict of the bromine analog being non-predictive of the iodine analog. " Claim 25. (New) A compound, Iodine acetamido benzoyl ethyl acetate, that causes a unique combination of effects when applied to cancer cells including causing S-phase arrest, biphasic bcl-2 phosphorylation and covalent modification of tubulin, which are not characteristics of its closest analog Bromine acetamido benzoyl ethyl acetate, and which result in anticancer properties. "

Clearly Claim 25 would be the point of reference for the statements in other claims which currently refer to Claim 1.

Point 3: Examiner's response p3 line 13 to p6 line 2.
We have amended the Claims 9 through 22 to be more descriptive.

Point 4: Examiner's response p6 line 3 to p8 line 2.
Since the last correspondence we have amended the status of the application to a continuation of USPTO # 6,294,695. In consideration of this fact, the Examiner's statements from Examiner's comments on 4/7/2004 (Page 6 paragraphs starting "Claims 8-21, 23 ... " and ending at the top of page 8) are moot because of the different characteristic of the iodine derivative from the bromine derivative. In particular as stated in Claim 25, "Iodine acetamido benzoyl ethyl acetate, that causes a unique combination of effects when applied to cancer cells including causing S-phase arrest, biphasic bcl-2 phosphorylation and covalent modification of tubulin, which are not characteristics of its closest analog Bromine acetamido benzoyl ethyl acetate ... ".
Also, the fact that the examiner assumed the current application was a continuation (see Examiner's correspondence 5-5-2004) is also moot because there is no text in the original application concerning a statement of continuation.
In addition there is a statement in the Parent Patent (p14, column 5, lines 30 to 34) that the Y substituent can be ethoxy or other groups thereby eliminating the anti-tubule activity.
Thus the compound is unique in its characteristics and the Examiner's objections are transversed, the Claims 8 though 23 are retained.

Point 5: Examiner's response p8 line 3 to p9 line 1

a) The dependency has been changed to Claim 25.
b) Medicinal drug adequately describes an anti-cancer drug, therefore the term is retained.
c) Medicinal drug as opposed to a Narcotic drug seems to be quite a good distinction, therefore the term is retained.
d) Capitalization denotes the real name of a particular disease, these capitals can be reduced to lower case if the Examiner deems it necessary.
e) The compound is in the meta configuration, Claims 23, 24 and 25 have been amended to reflect this.

Point 6: Examiner's response p9 line 2 to p10 line 7.
The Examiner's statements are moot because of the different characteristic of the iodine urea derivative of Jiang et al 1998. In particular as stated in Claim 25, "Iodine acetamido benzoyl ethyl acetate, that causes a unique combination of effects when applied to cancer cells including causing S-phase arrest, biphasic bcl-2 phosphorylation and covalent modification of tubulin, which are not characteristics of its

closest analog Bromine acetamido benzoyl ethyl acetate ... ", and these characteristics are not characteristics of its closest urea analog.

Thus the compound is unique in its characteristics and the Examiner's objections are transversed, the Claims 8 though 23 are retained.

Point 7: Examiner's response p10 line 8 to p17 line 5.
The Examiner's previous statements reflected a view of a rigid cell cycle (e.g. 6-23-2003 p.12 first paragraph), now the Examiner encompasses the idea that the cell cycle is quite flexible. We appreciate the descriptions of cell cycle but for this application it is the combination of tubulin ligand, S-phase arrest, biphasic phosphorylation and tubulin covalent modification which characterize this unique compound, these aspects are described in Claim 25.

Points of concern:

Point 8: Claiming a compound and a mechanism is possible.
As concerns the mechanism of action, and in addition to fax correspondence on 1-1-2004, we note: USPTO # 6660767. In the inventors' claims it clearly states that "stabilizing microtubules" is the mechanism to be claimed. Likewise we are claiming that "S-phase arrest" is the mechanism. Thus the rejections are transversed and Claim 8 is retained.
In addition in USPTO # 6660767 the inventors clearly claim a compound "coumarin" without a method of synthesis. Likewise we are claiming a compound "iodine acetamido benzoyl ethyl acetate" without a method of synthesis. The method of synthesis is clearly stated in USPTO # 6,294,695, of which the current application is a continuation. Thus the rejections are transversed and Claims 23 and 24 are retained and a new Claim 25 is added.

Point 9: Parity between recently allowed patents and the treatment of this application.
We are still looking for parity between our application and other approved patents, especially #6660767. The Examiner's response (mailed 5-5-2004) to the inventors submittal (1-1-2004) was to dismiss the letter entirely as irrelevant. The inventors submittal is highly relevant to the main concerns of the Examiner, namely the patentability of a compound and a mechanism. We ask the Examiner to respect the inventors request for due process, that a full response to the inequality between #6660767 and the current application's status is written in the response to this letter.

Point 10: The current status of all Claims is as follows:

Claims

Claim 1 [Canceled]

Claim 2. [Canceled]

Claim 3. [Withdrawn, see Claims 8-21] Tubulin ligands with a G1/S-phase arrest mechanism of anti-cancer activity.

Claim 4. A tubulin ligand that causes a biphasic phosphorylation of bcl-2, one phase being between 1-3h and the next phase being 12h onwards.

Claim 5. [Canceled]

Claim 6. [Canceled]

Claim 7. [Canceled]

Clam 8: A tubulin ligand that causes a G1/S-phase arrest.

Claim 9: (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes tumor volume reduction of prostate cancer in a mouse model organism.

Claim 10: (currently amended) The medicinal drug of Claim {[1]} 25 wherein it cures prostate cancer in 20% of cases in a mouse model organism.

Claim 11 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of T-cell Leukemia cancer cells at a lower dose (0.047ug/ml) than the closest available control cells Normal Lymphocytes (2.5ug/ml).

Claim 12 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of a Myelodisplasia syndromes cell type such as the Sp cell line at much lower doses (0.085ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 13 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of a Melanoma cell type (DND-1A) at much lower doses (0.25ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 14 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it has anti-cancer activity.

Claim 15 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of Renal Cancer cells (cell line 786-0) at much lower doses (0.042ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 16 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of Breast Cancer cells (cell line MCF-7) at much lower doses (0.12ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 17 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of Non-small Cell Lung Cancer cells (cell line NCI-H522) at much lower doses (0.05ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 18 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of Colon Cancer cells (cell line HCT-116) at much lower doses (0.09ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 19 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of lymphoma ~~wild type~~ cancer cells (cell line Daudi / MDR) at much lower doses (0.007ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 20 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it causes ~~selective~~ cell death of lymphoma MDR negative cancer cells (cell line Daudi-MDR) at much

lower doses (0.005ug/ml) than the nearest control cell line Normal Lymphocytes (2.5ug/ml).

Claim 21 (currently amended) The medicinal drug of Claim {[1]} 25 wherein it does not cause cell death of normal lymphocytes when added to a culture of these cells at the ID90 dose of lymphoma e.g. 0.1ug/ml.

Claim 22 A situation where the 1-3h Bcl2 phosphorylation phase could be used as a tool to develop anti-cancer drugs with similar or improved potency.

Claim 23 (currently amended) One novel compound: Iodine meta-acetamido benzoyl ethyl acetate.

Claim 24. Halogenated meta-acetamido benzoyl ethyl acetate derivatives that covalently bind to tubulin.

Claim 25. (New) A compound, iodine meta-acetamido benzoyl ethyl acetate, that causes a unique combination of effects when applied to cancer cells including causing S-phase arrest, biphasic bcl-2 phosphorylation and covalent modification of tubulin, which are not characteristics of its closest analog Bromine acetamido benzoyl ethyl acetate, and which result in anticancer properties.

Yours sincerely,

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